that suggested that pancreatic ascites was caused by a pancreatic duct disruption, other theories erroneously proposed that blocked intraperitoneal lymphatics or subacute peritoneal inflammation, or both, were causes of pancreatic ascites. The irritation of the peritoneum by pancreatic secretions results in the high albumin levels noted in pancreatic ascitic fluid; albumin is *not* secreted by the pancreas. However, with the increasing use of retrograde pancreatography in evaluating patients with pancreaticobiliary disease, it has been shown unequivocally that pancreatic ascites is caused by a tear in a major pancreatic duct or from disruption of an incompletely formed pseudocyst.[2,6] In the case presented above, a fistulous tract from one of two pancreatic pseudocysts located in the head was clearly found to be emptying into the peritoneal cavity.

In addition to the complication of pancreatic ascites, our patient also had transenteric rupture of a pseudocyst into the postbulbar duodenum. This was recorded by the endoscopic visualization of a hole in the duodenal wall that, when cannulated and injected with a contrast agent, was shown to be a pseudocyst with a connecting fistulous tract. Transenteric cyst rupture can occur in any part of the adjacent gastrointestinal tract, with one series showing an equal predilection for the stomach, duodenum and colon. Rarely, pseudocysts have been noted to rupture into the esophagus or small bowel.[3]

Although transenteric rupture of pancreatic pseudocysts may result in resolution of the cysts, this occurrence is not without its hazards. Half of these events are associated with gastrointestinal hemorrhage and, in one series, all seven deaths complicating cystoenteric rupture were attributed to massive bleeding.[3,7] Presumably this hemorrhage is the result of through-and-through erosion of the bowel wall caused by the adjacent pseudocyst or erosion into nearby vessels during the process, or both.

Such may actually have been the cause of death in our patient. Although no ulcer was seen endoscopically at the time of either ERCP, a barium study showed a "probable" ulcer in the duodenal bulb. Closer inspection of these films, however, showed air in what could very well have been the remnants of the second pseudocyst, which had ruptured into the duodenal bulb (the first one rupturing into the postbulbar duodenum). Supporting this hypothesis is the fact that the limited autopsy, albeit a superficial one, failed to show any residual pancreatic pseudocysts and that the crater in the bulbar area was adherent to the pancreatic head. Furthermore, a recent case report described the findings of a barium study that suggested a duodenal ulcer, which subsequent endoscopy and fistulography confirmed to be a tract from the duodenal bulb into a pancreatic pseudocyst.[8]

The vascular anatomy in the region of the pancreatic head and duodenal bulb is such that it would not be at all difficult to implicate an acute erosion into a branch of the gastroduodenal artery to account for the rapid exsanguination in our patient.

A reasonable approach to the treatment of pancreatic ascites would be to attempt medical therapy for several weeks. Central parenteral hyperalimentation will "rest the pancreas" and perhaps allow a fistula to close spontaneously; some physicians successfully treated pancreatic ascites with irradiation.[9] In these cases it is theorized that low-dose irradiation (550 rads) did not induce fibrosis but rather an acute, reversible diminution in enzyme secretion that contributed to healing of the ductular tear or decreased the leakage from a pseudocyst.

Most patients with pancreatic ascites will not respond to medical therapy but will at least have had improvement of their nutritional status before the major surgical procedure required. The success of surgical intervention is clearly related to accurately locating the leak, as Sankaran and colleagues reported a recurrence rate of 53% in 19 patients who had no pancreatogram, but no recurrences in 7 patients with endoscopic localization.[10] ERCP constitutes state-of-the-art preoperative diagnostic management in these patients, and although it uncommonly shows the leak as clearly as in the case presented here, it will define the ductular anatomy, its relationship to pseudocysts, if any, and afford surgeons a road map before they embark on a perilous surgical journey.

REFERENCES

1. Cameron JL, Kieffer RS, Anderson WJ, et al: Internal pancreatic fistulas: Pancreatic ascites and pleural effusions. Ann Surg 1976 Nov; 184:587-593
2. Rawlings W, Bynum TE, Pasternak G: Pancreatic ascites: Diagnosis of leakage by endoscopic pancreatography. Surgery 1977 Mar; 81:363-365
3. Bradley EL III, Clements JL Jr: Transenteric rupture of pancreatic pseudocysts: Management of pseudocystenteric fistulas. Am Surg 1976 Nov; 42:827-830
4. Cameron JL: Chronic pancreatic ascites and pancreatic pleural effusions. Gastroenterology 1978 Jan; 74:134-140
5. Cameron JL, Anderson RP, Zuidema GD: Pancreatic ascites. Surg Gynecol Obstet 1967 Aug: 125:328-332
6. Sankaran S, Walt AJ: Pancreatic ascites: Recognition and management. Arch Surg 1976 Apr; 111:430-434
7. Wolstenholme JT: Major gastrointestinal hemorrhage associated with pancreatic pseudocysts. Am J Surg 1974 Apr; 127:377-379
8. Hoff G, Heldaas J: Pancreatic pseudocyst and pancreatico-duodenal fistula diagnosed by endoscopic fistulography. Endoscopy 1981 Sep; 13:221-222
9. Morton RE, Deluca R, Reisman TN, et al: Pancreatic ascites: Successful treatment with pancreatic radiation. Am J Dig Dis 1976 Apr; 21:333-336
10. Sankaran S, Sugawa C, Walt AJ: Value of endoscopic retrograde pancreatography in pancreatic ascites. Surg Gynecol Obstet 1979 Feb; 148:185-192

Maggot Therapy Revisited

JURIS BUNKIS, MD
Hayward, California

SCOTT GHERINI, MD
San Francisco

ROBERT L. WALTON, MD
Worchester, Massachusetts

THE BENEFICIAL EFFECT of maggot therapy on wound debridement has been known to surgeons since ancient times.[1] Baer popularized this method when he reported in 1931 the successful treatment of 89 cases of intractable osteomyelitis by the application of blowfly larvae.[2] Maggot therapy flourished during the next ten years, but its popularity soon waned with the introduction of antibiotics and antiseptic solutions. Today, the intentional use of maggots for the treatment of chronic wounds has been nearly abandoned.

In this report we describe a case of a patient with a ne-

(Bunkis J, Gherini S, Walton RL: Maggot therapy revisited. West J Med 1985 Apr; 142:554-556)

From San Francisco General Hospital Medical Center and the Division of Plastic and Reconstructive Surgery, Department of Surgery, University of California, San Francisco, School of Medicine. Dr Walton is now Associate Professor and Chair, Plastic Surgery Division, Department of Surgery, University of Massachusetts Medical School, Worcester.

Submitted January 25, 1984.

Reprint requests to Juris Bunkis, MD, 1320 Apple Ave, Suite 104, Hayward, CA 94541.

crotic facial tumor that was infested with maggot larvae when the patient first presented for treatment. The larvae were left in situ and successfully debrided the wound before surgical therapy.

Report of a Case

The patient, an 88-year-old woman, was referred from a local convalescent hospital to the Emergency Room of San Francisco General Hospital Medical Center with a chief complaint of maggot invasion of a facial tumor. The patient did not recall the duration of the lesion and, due to poor vision, was unaware of the presence of the larvae. She said she did not have facial pain, pruritus or fever. On examination she had a 4×4-cm raised, necrotic ulcer over the right upper cheek. The wound contained several blowfly larvae; there was no evidence of surrounding cellulitis or palpable adenopathy. Her overall hygiene was otherwise normal and physical examination showed no abnormalities. A tentative diagnosis of squamous cell carcinoma was made and confirmed by an incisional biopsy.

In light of the innocuous appearance of the wound and an agreeable patient, we elected to continue supervised wound care using resident maggots (Figure 1). The exact species of the blowfly larva was not determined. The wound was covered with a sterile moist saline dressing and observed several times a day. By the third day the wound was found to be clean and void of any necrotic residua. The remaining maggots were then removed by topically applying ether.

One week after admission, the facial lesion remained free of necrotic tissue and larvae. Cultures of surface wound showed a scant growth of contaminating bacteria. The lesion was excised and closed with a local transposition flap. Postoperative recovery was unremarkable and the patient returned to her nursing home.

Discussion

Baer's 1931 report describing the use of blowfly larvae for the treatment of chronic osteomyelitis heralded a decade of investigation into the physiologic basis of maggot therapy.[2] One must recall that during this period the mortality for compound lower extremity fracture was 75%, mainly due to the complications of osteomyelitis. In his series of 89 patients with chronic osteomyelitis, Baer reported a success rate of 90% using maggot therapy—especially remarkable considering that some of his cases involved tuberculous osteomyelitis and other intractable lesions. During the 1930s and early 1940s, maggot therapy was used successfully for a variety of other suppurative infections including abscesses, burns, gangrene and chronic leg ulcers. Stewart defined the ideal lesion to treat with maggot therapy: a shallow wound with a relatively large amount of necrotic tissue.[3] Robinson found that the number of maggots per wound varied from 200 to 600, which consumed an average of 10 to 15 grams of necrotic tissue a day.[4,5]

The life cycle of the maggot, larva of one of several blowfly species of the Calliphoridae family, has been studied by Baer and others.[2,3,6,7] Baer found that maggots could be secured by exposing raw beef outdoors on which flies would deposit their eggs. Blowflies will also deposit eggs in open wounds or parts of dead birds and animals not covered by feathers or hair, and occasionally on feces. The eggs hatch in 8 to 24 hours and the larvae begin feeding. The newly hatched maggots secrete a proteolytic enzyme capable of extracorporeal digestion, following which the liquefied food is swallowed into the alimentary tract and perhaps absorbed through the integument. Within a week or two, the larvae convert into pupae, and this is followed by the emergence of flies a few days later.

Early attempts at maggot therapy were occasionally followed by the unfortunate appearance of anaerobic infections. Of these, *Clostridium perfringens* and *Clostridium tetani* infections were most worrisome. Attempts to sterilize maggots by soaking them in hydrogen peroxide were unsuccessful. Although the bacteria could be initially eliminated from the surfaces of the larvae, endogenous intestinal tract bacteria survived and soon recolonized externally. Baer discovered, however, that the surfaces of unhatched fly eggs could be successfully sterilized by soaking them in a bichloride solution at room temperature for one hour. This technique produced sterile larvae that could then be reared in a sterile environment until they were applied to a wound. The potential for causing a nosocomial infection was thus eliminated.

The success of maggot therapy is not limited to their scavenger action. Robinson suggested that maggot products—allantoin, urea and, most important, ammonium bicarbonate—have considerable healing properties.[8] Other proposed benefits of maggot therapy, most of which remain unproved, have included rinsing out and dilution of bacteria in a wound as a result of the serous exudate induced by the presence of maggots, enhancement of granulation tissue formation by the continuous crawling of the larvae, enzymatic liquefaction of necrotic tissue, destruction of wound bacteria in the alimentary tracts of the maggots, the use of necrotic tissue for food by larvae, the secretion of antibacterial agents by the larvae, increased wound alkalinity and the release of various other agents supposedly beneficial to wound healing (such as calcium picurate, sulfhydryl, amino acids and the like).[5,6,9-11] Vistnes and co-workers have suggested that a suitable form of

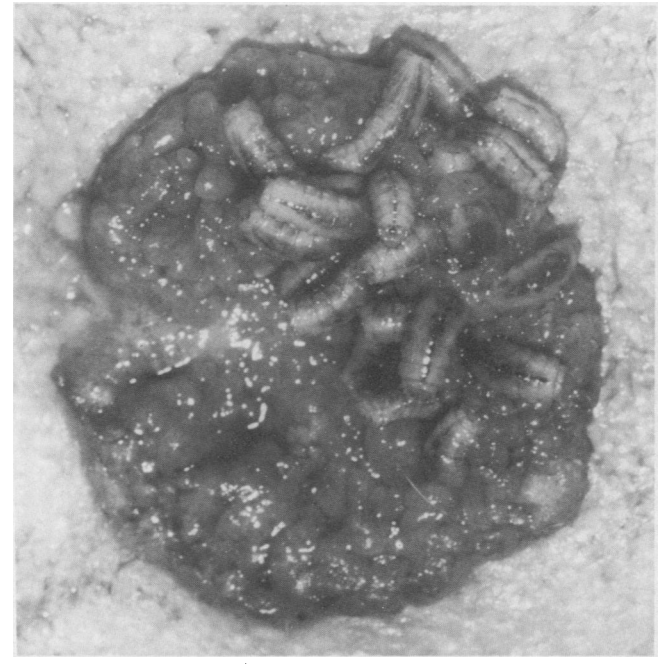

Figure 1.—Full-grown larvae present in a cheek wound of an 88-year-old woman on the second hospital day.

maggot proteolytic secretions could be useful for enzymatic wound debridement.[12]

Maggot therapy has certain undeniable advantages: blowfly larvae consume only necrotic tissue, thereby leaving vital structures unscathed; maggots act topically and are not reliant on an excellent peripheral blood supply; maggots are universally available, and maggot therapy is outstandingly cost-effective.

The chief disadvantage of maggot therapy is an aesthetic one. In this post-Listerian era, it is difficult to convince hospital staff and patients of the potential benefits of maggot therapy. Another possible disadvantage of maggot use is the incitement of an intense local pruritus (noted by earlier investigators). Our patient, however, was oblivious to the maggots' presence—a factor that allowed us to continue their use until the wound was debrided.

The dawning of the antibiotic era effectively brought a halt to the popularity of maggot use for wound debridement. Due to social disapproval, it is unlikely that maggot therapy will ever regain the level of popularity it enjoyed in the 1930s and 1940s. As recently as 1976, however, Horn and associates showed that maggots may be used successfully to treat a case of subacute mastoiditis unresponsive to conventional therapy.[13]

Despite a rather extensive armamentarium in wound therapy, contemporary physicians occasionally encounter a special sore in a special patient in a special circumstance that dictates special care. While not advocating a step backward in time, we feel that this unique, albeit unconventional, approach to wound management may occasionally deserve consideration.

REFERENCES

1. Pechter EA, Sherman RA: Maggot therapy: The surgical metamorphosis. Plast Reconstr Surg 1983 Oct; 72:567-570

2. Baer WS: The treatment of chronic osteomyelitis with the maggot (larva of the blow fly). J Bone Joint Surg 1931 Jul; 13:438-475

3. Stewart MA: A new treatment of osteomyelitis. Surg Gynecol Obstet 1934 Feb; 58:155-165

4. Robinson W: The use of blowfly larvae in the treatment of infected wounds. Ann Entomol Soc Am 1933 Mar; 26:270-276

5. Robinson W, Norwood VH: The role of surgical maggots in the disinfection of osteomyelitis and infected wound. J Bone Joint Surg 1933 Apr; 15:409-412

6. Baer WS: Viable antisepsis in chronic osteomyelitis. Proc Interstate Postgrad Med Assem North Am 1929 Oct; 5:365-372

7. Fabre JH: The Life of the Fly. New York, Dodd, Mead, 1920

8. Robinson W: Ammonium bicarbonate secreted by surgical maggots stimulates healing in purulent wounds. Am J Surg 1940 Jan; 47:111-115

9. Livingston SK: The therapeutic active principle of maggots. J Bone Joint Surg 1936 Jul; 18:751-756

10. Robinson W, Baker FC: The enzyme urease and occurrence of ammonia in maggot infected wounds. J Parasitol 1939 Apr; 25:149-155

11. Simmons SW: Stimulation of healing in nonhealing wound by allantoin occurring in maggot secretions and of wide biological distribution. J Bone Joint Surg 1935 Apr; 17:267-271

12. Vistnes LM, Lee R, Ksander GA: Proteolytic activity of blowfly larvae secretions in experimental burns. Surgery 1981 Nov; 90:825-841

13. Horn KL, Cobb AH Jr, Gates GA: Maggot therapy for subacute mastoiditis. Arch Otolaryngol 1976 Jun; 102:377-379

AACIA CASE HISTORY CONTEST

The American Association of Clinical Immunologists and Allergists will be starting a new section in its journal, *Immunology and Allergy Practice,* entitled "The Case History of the Month."

In order to generate interest in this new feature, Searle Pharmaceutical Company will be sponsoring a contest consisting of a $300 prize for the best paper in each of the following categories:

1. A physician in private practice,
2. A physician in academics,
3. A physician in training.

These case reports may be in any field relating to allergy, clinical immunology, obstructive pulmonary disorders or related ear, nose and throat problems.

An original and two copies of each paper should be sent, along with the category in which the physician would like to be considered. The contest deadline is December 31, 1985. Correspondence should be addressed to:

GERALD L. KLEIN, MD
161 Thunder Drive, Suite 210
Vista, CA 92083